

NERVOUS REGULATION OF THE CIRCULATION AND RAPID CONTROL OF ARTERIAL PRESSURE

AP α CO α TPR

AP = arterial pressure

CO = cardiac output

TPR = total peripheral resistance

The nervous system controls the circulation, specially autonomic nervous system (sympathetic nervous system).

Sympathetic inervation to : 1) all blood vessels except capillaries. 2)the heart.

Parasympathetic "vagal inervation " mainly to the heart.

When there is sympathetic stimulation for arteriols and small arteries cause :

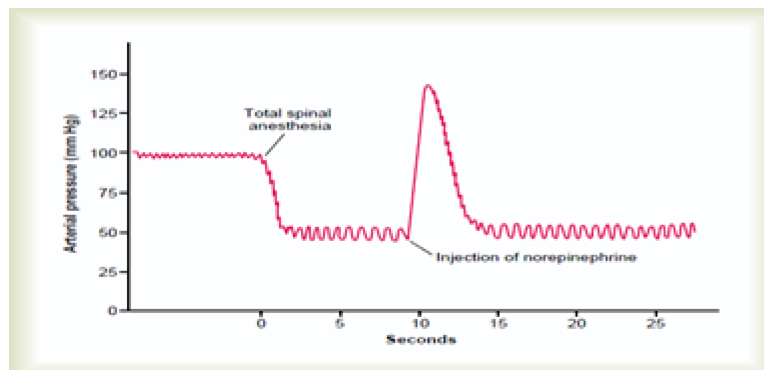
1) Vasoconstriction : means that resistance will increase (AP α CO α TPR) consequently arterial pressure will increase .

2) Increase in heart rate → increase force of contraction→ increase cardiac output→ increase AP.

- Parasympathetic stimulation to the heart will decrease the heart rate → decrease force of contraction → the cardiac output will decrease → decrease in AP.

In the brain stem, there is very important center called VASOMOTOR CENTER. The vasoconstrictor area of the vasomotor center normally transmits continuously signals to the sympathetic vasoconstrictor nerve fibers throughout the body. These fibers will make slow continuous discharge called SYMPATHETIC VASOCONSTRICTOR TONE. The benefit of this tone is to maintain partial state of contraction in the blood vessels, this state is called VASOMOTOR TONE. The sympathetic vasoconstrictor tone is important to maintain normal blood pressure (normal arterial pressure).

The evidence of this is an experiment done on an animal , when they did total spinal anesthesia to it. This anesthesia blocked the transmission of sympathetic impulses from spinal cord to the peripheral organs, so the arterial pressure decreased to zero.



When some people face emotional shock, they faint. This is called VASOVAGAL SYNCOPE or EMOTIONAL FAINTING. Because this intense emotional disturbance, signals pass into cerebral cortex to the anterior hypothalamus and from it signals pass into the vagal center of the medulla and from it signals pass in two pathways :

1)To the heart : cause decrease in heart rate and force of contraction so decrease in cardiac pressure and arterial pressure.

2)Through the spinal cord to the sympathetic vasodilator center : cause vasodilation → cause decrease in resistance.

So both pathways cause decrease in AP so decrease blood flow to the brain which causes fainting or loss of consciousness.

When the sympathetic stimulation occurs signals not only pass to blood vessels but also the adrenal medulla which secret two hormones: epinephren 80% and 20% norepinephren.

1) Norepinephren can cause : vasoconstriction and increase heart rate and force of contraction because it has strong effect on alpha receptors.

2)Epinephren : has weak effect on alpha receptors but it affects more on beta receptors so it causes weak vasoconstriction , sometimes causes vasodilation of certain vessels in few tissues, in addition to its effect on the heart which can increase the heart rate and the force of contraction.

When the sympathetic stimulation occurs there is dual effect :

* by blood vessels (direct inervation cause vasoconstriction which increases the resistance).

* by affecting on the adrenal medulla to secret its two hormones that do vasoconstriction and increase the heart rate and the force of contraction, so they can cause increase in blood pressure.

Concerning the rapid control of the AP, the nervous system has the ability to rapidly control the AP "within seconds ".

1. The first nervous reflex concerning nervous control of AP is called BARORECEPTOR REFLEX.

The baroreceptors are stretch receptors present on the wall of the arteries in the thorax (chest) and neck regions but present in a higher level on the wall of the aorta AORTIC BARORECEPTORS and the wall of each internal carotid artery called CAROTID SINUS.

when the AP increases these baroreceptors stretch and send signals to a sensory area located in the tractus solitarius in the brain stem and this sensory area sends

1) inhibitory signals to the vasoconstrictor area of the vasomotor center: will cause vasodilation.

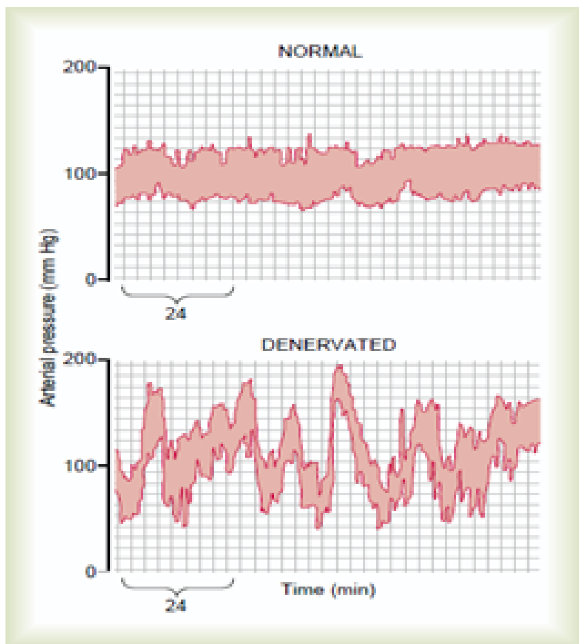
2) excitatory signals to the vagal center: will lead to decreased heart rate and the force of contraction so cardiac output will decrease and arterial pressure will decrease toward normal.

when AP is lower than normal, these baroreceptors become inactive (No impulse).This means that normally, even when the AP is normal there is a resting level of discharge from the baroreceptors to the vasoconstrictor area of the vasomotor center and vagal center which make blood pressure normal. So when AP decrease the action of the vasomotor center will increase because of the absence of the normal effect on it.

No active baroreceptors→ no impulses from them→ no inhibitory signals on the vasomotor center and no excitatory signals on the vagal center → so the vasomotor center become more active than normal → so the vasoconstriction will increase and the heart rate and the force of contraction will increase so the AP will increase toward normal.

There is effect of baroreceptors even in change of body posture. When the person is laying down and suddenly stands up there will be tendency of decrease of AP in the upper part of the body (the head and upper parts) so the person will have fainting but because of the baroreceptor reflex this is rapidly corrected.

It is found to act in simple daily events. If there is denervation of the baroreceptors, there will be extreme variability of the mean arterial pressure during the day, An experiment was done on animals .



Simple events of the day can be : noise, excitement , suddenly standing , defecation can change AP but when the baroreceptors are functioning normally so there will be no problem and the AP won't change significantly. So the baroreceptor reflex helps to maintain AP when there is change in it (increase or decrease) and it also participate in maintaining normal arterial pressure during the day and when exposing to simple daily events.

Are the baroreceptors important in long term regulation of the AP?

In general, NO. Because when AP increases baroreceptors send signals but gradually after a day or two these impulses decrease toward normal even if the pressure is still high. In this case, it is not considered as long term control.

Even when AP decreases baroreceptors are inactivated but also after a day or two it comes back to normal " this is called ADAPTATION ". So we can say that , in general the baroreceptors are not important for long term regulation of arterial pressure.

But some experimental studies state that the baroreceptors doesn't show complete adaptation and they may contribute to long term regulation. By decreasing the sympathetic stimulation to renal vessels and if this happened the glomerular capillary pressure increase causing increase in filtration of fluid through renal tubules into the urine so more loss of fluid that can lead to decrease extracellular fluid volume → decrease in blood volume → decrease in cardiac output toward normal.

2. other reflex is caused by other receptors called CHEMORECEPTOR REFLEX.

The chemoreceptors are chemosensitive cells located in the aortic arch "AORTIC BODIES" and the bifurcation of the common carotid arteries " CAROTID BODIES" these chemosensitive cells or

chemoreceptors in this bodies are sensitive to the deficiency of oxygen or excess of hydrogen ions and carbon dioxide. these bodies receive rich blood supply by small nutrient artery.

When mean AP decrease below 80mmHg these chemoreeptors are stimulated and:

1) send their impulses to the vasomotor center through nerve fibers causing its excitation which will try to increase the AP to normal by the previously described mechanisms.

2)signals are also transmitted through skeletal nerves to skeletal muscles of the body especially the abdominal muscles so their contraction increases " increase their muscular tone "→ cause compression of the venous reservoirs of the abdomen such as spleen , liver and large abdominal veins so there will be translocation of the blood toward the heart which helps increasing venous return→ increae cardiac output → increase AP toward normal. This is called ABDOMINAL COMPRESSION REFLEX.

3. there are stretch receptors in both atria and pulmonary arteries called LOW PRESSUR RECEPTORS, because they are located in places where the pressure is low " atria and pulmonary arteries.

When there is increase in blood pressure due to increased volume (because these receptors are not located in the systemic circulation, they can't detect AP) this low pressure receptors are stimulated so they send signals to : 1) kidneys.2) hypothalamus . To do reflex dilation of the afferent arterioles of the kidney → increase in the glomerular capillary pressure so the filtration of the fluid into renal tubules will increase. Signals that go to the hypothalamus decrease ADH secretion (ADH is formed in the hypothalamus but secreted from the posterior pituitary gland) . ADH stimulates re-absorption of fluid, so decreased ADH secretion cause decrease in re-absorption of fluid ,or in other words, increase excretion of fluid.

So both effects on the kidney and the hypothalamus will cause increase excretion of fluid by the kidneys so decrease extracellular fluid volume toward normal → decrease blood volume toward normal → decrease cardiac output toward normal so decrease AP toward normal.

Also when there is stretch in the atria by increase in blood pressure due to increased volume, atria release a factor called ATRIAL NATRIURITIC PEPTIDE , which increase excretion of sodium and water and help in loss of excess fluid so decrease extra cellular fluid volume, blood volume, cardiac output and AP toward normal.

4. CENTRAL NERVOUS SYSTEM ISCHEMIC RESPONSE :

When the mean AP decrease to 60mmHg or below, the blood flow to the vasomotor center will decrease much leading to what is called cerebral ischemia . In this case, there will be deficiency of O_2 and excess of CO_2 and other acidic substances like lactic acid , so these substances cause strong stimulation of neurons in the vasomotor center so it will lead to increase the AP toward normal.

For other figures look at guyton chapter 18

هذا عمل طلابي قد لايخلو من الخطأ فالرجاء اعلامنا في حالة وجود خطأ . بالتوفيق